being required for Notch signaling in the pIIa cell. In agreement with an additional, Numb-independent function for Spdo in the endocytosis of Notch, the amount of NiGFP was increased in *spdo* mutant SOPs compared with wild-type SOPs or *numb*-mutant SOPs [6]. Antibody internalization experiments and proximity ligation experiments, which indicated the formation of a protein complex between Spdo and Notch, point to the co-internalization of Notch and Spdo from the plasma membrane. Notably, in contrast to SOPs lacking Numb, NiGFP remained undetectable at the basal side of the cytokinetic furrow in SOPs that lack expression of Spdo or of both Spdo and Numb. This suggests that, in pIIb cells, Spdo-dependent and Numb-independent endocytosis is required to transport Notch to the basal side of the cytokinetic furrow and that the turnover of Notch at this site is mediated by a Numb- and dynamin-dependent endocytic process. The finding that apparently no NiGFP accumulated at the basal interface between pIIb and pIIa cells during normal SOP development additionally suggests that pIIa cells are able to remove Notch from the basal side of the cytokinetic furrow in a Numb-independent manner. Alternatively, Notch might be trafficked differently in wild-type pIIa cells compared to *numb*-mutant or *numb*-silenced pIIb cells, maybe via routes that do not involve the basal side of the cytokinetic furrow.

In conclusion, these results provide the first *in vivo* evidence for a function of Numb in the endocytosis of Notch. They furthermore demonstrate that at least two partially interdependent endocytic pathways control Notch subcellular localization and trafficking during asymmetric cell division, introducing a new level of complexity in the regulation of cell-fate decisions.

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Evolution: Why Good Dads Win

Males usually do not provide parental care and with good reason, they may be caring for the offspring of someone else. But there are cases of male-only care even when certainty of paternity is low: why? A new model suggests female choice may provide the answer.

Tom A. Price[1] and David J. Hosken[2]

Pairs of birds happily building nests together and feeding their young is a staple of schmaltz, from Disney to nursery rhymes. But the evolutionary reasons for this shared parental care, and especially the male care, puzzle behavioural ecologists. Male care is simple to understand when males and females form monogamous pairs: the offspring share 50% of the father's genes and his care can increase their success [1]. However, females of most species are not monogamous and they typically mate with several males. Hence the male that cares for the young, the 'social partner', may only be the father of some, or indeed none, of the offspring produced by his partner. This uncertainty of paternity is thought to be one reason why females care more than males [2] — they know the kids are theirs, while males are never sure. Variation in male mating success only exacerbates this problem. On the one hand, attractive males that are good at securing matings are not expected to provide care for offspring because they can do better by spending their time mating with additional females. Conversely, unattractive males should not care for offspring because they will probably not have fathered them. Thus the overwhelming pattern across the animal kingdom is that females provide care more often than males [3,4].

Despite good reasons for not caring, and in stark contrast to the general pattern of female-only care, males of some species nonetheless do provide parental care, sometimes when cues indicate the offspring are not theirs [5],

and sometimes males are the sole carers. Indeed, in some species males appear to increase care when more of the offspring are not theirs [6,7]. Current theory suggests that male parental care is maintained through strong natural selection: offspring survival is very low without male care, so it is still worth caring, even if few of the offspring cared for are fathered by the caring male. But although sole care by males occurs even when certainty of paternity is low and sexual selection is acting on males, and male-only care is found across a broad taxonomic distribution, there has been little investigation of why this might be. A new model by Suzanne Alonzo [8] sheds some light on this puzzle.

Alonzo [8] investigates what happens when females show a preference for males who are more likely to care for offspring. In her model, females always gain increased fitness by mating with a male who is more likely to care. Hence females are expected to evolve increased preference for more caring males. This creates a situation where females continually evolve a stronger preference for more caring males, which in turn means that, no matter how costly care is to males in terms of reduced mating success, eventually the female preference for caring males will be larger. At this point, the benefits of care outweigh the costs, and increased male care should evolve. Unlike previous models, even those that suggest female preference could counter the costs of care [9], this model can explain the evolution of uniparental care by males, because female preference for caring males can potentially be so important to male fitness that caring is worthwhile even when the female deserts him and the offspring.

In some ways these outcomes resemble Fisherian predictions [10] — when female preference is strong enough it can drive the evolution of male traits beyond their naturally selected optima (here, no care) — and in others, it reflects a prediction made by Seger and Trivers [11] — that female preference for a male signal which indicates how good the male would be as a female (here, how caring a male is) could evolve more rapidly than signals of masculine quality. At one level, however, the model predictions are trivial — with strong enough selection for male care, caring males will evolve. What is non-trivial is that the model predicts male care should evolve easily and, surprisingly, certainty of paternity has little impact on the evolution of male care as long as females prefer and can select caring males to sire their offspring. Thus while investigating situations where we have male only care, the model ends up posing the more interesting question: why don't we find more situations where males are the sole carers?

The answers provided by the model are that male care should not evolve: when female preference for caring males is not favoured by selection; when males do not express traits that enable females to discriminate between caring and non-caring males; when something limits females' ability to bias paternity toward caring males; when the genetic architecture of the species limits the evolution of either female preference or male trait, or there is a lack of positive genetic covariance between the two.

Some of these explanations are deeper than others. For example, genetic architecture can evolve [12], so an absolute genetic constraint may be unimportant over evolutionary time, and lack of selection for care preference is in some ways rather trite. However, why males may not reveal their quality as carers is very interesting and two strong predictions emerge from the above to explain cases of care or its lack. Firstly, females must be able to identify which males will care for offspring. And secondly, females must be able to bias paternity towards these caring males. Hence, males in species with male care should show some honest signal of care. Furthermore, females must have a high degree of control over paternity if males that put high effort in matings or gains per mating (non-caring males) are to be out-competed by caring males with fewer resources to invest in mating attempts.

In some species, both conditions are likely to be found. In many fish that show male care, females assess males by examining the nests they have built, and looking for the presence of well tended eggs from other females [13,14]. In these species, biasing paternity towards these males is simple: the female simply deposits a greater quantity of her eggs with these males. In internally fertilising species, however, significant paternity biasing is probably more complicated [15]. Nevertheless, there is at least some evidence that females may increase the paternity of males who care well for their young [7].

The model's assumptions may provide additional explanations for a general lack of male care in nature despite the ease at which it emerges from the model. For example, there is no cost to female choice in the model. This was an initial criticism of Lande's model of the Fisher process [16], but recent work has shown that criticisms may have been misplaced [17]. Additionally, the Alonzo model [8] was built with fish and birds in mind, and in systems such as sedge warblers where song provides an honest signal of male care [18], the costs of assessment are likely to be low. Another cost that may have more importance is the cost of care itself. Currently the model assumes no survival cost to care and importantly no sex differences in survival. Male survival is frequently lower than female survival, often because of costly sexual selection, including costly revealing traits. If this makes care more costly for males, then the threshold strength of female preference needed for caring-male evolution may be greater than it appears, potentially reducing the parameter space under which we expect to find male care.

The model also assumes males either providing care or not, a simple binary choice, with males rated by the percentage likelihood they will provide care. This creates a situation where females will always gain fitness by choosing a male that is more likely to care. However, in many species, caring males also differ in the amount of effort they put into offspring, and offspring survival may not increase linearly with male care. If there are diminishing returns from male care, then the probability of an equilibrium at a lower level of male care, or no male care at all, would seem to increase.

Despite these caveats, Alonso's model [8] provides an interesting new perspective on why males might care for offspring not necessarily their own, and again highlights the fundamental importance of female preference in sexual selection. The predictions Alonzo generates should stimulate new empirical work, and in the end, the most interesting question might be if females can choose caring males, why is male care not more common? Deadbeat dads might end up being harder to explain than caring ones.

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Wnt Signaling: The Many Interfaces of β-Catenin

Wnt signaling regulates virtually every cell fate decision during development. How can the same signal trigger such diverse events? Engaging different transcriptional machinery via different protein interfaces on the transcriptional co-activator β-catenin provides part of the answer.

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A small subset of animal signaling pathways play inordinately important roles in development, with their transcriptional outputs shaping virtually every cell fate decision. Strikingly, inappropriate activation of these same powerful signaling pathways is implicated in most solid tumors. A central mystery in development is how the same signaling pathway directs diverse cell decisions. For example, Wnt signaling, one key pathway, modulates decisions as temporally and spatially diverse as dorsal–ventral axis formation, patterning of the mid/hindbrain, and adult bone mass homeostasis [1]. How can one pathway regulate the radically different sets of target genes required?

To answer this question, we must focus on the transcriptional Wnt effector, β-catenin (βcat). In the absence of Wnt signals, βcat is targeted for destruction [2], keeping intracellular βcat levels low, and Wnt target genes are repressed by TCF/LEF family DNA-binding proteins and their co-repressor Groucho. Wnt ligands inactivate the destruction complex, βcat levels rise and it outcompetes Groucho for TCF/LEF binding, driving expression of Wnt target genes. However, βcat has a dual life in a different cellular location, as part of the cadherin–catenin complex that is key to cell adhesion [3]. As a result, studying βcat's role in Wnt signaling and cell fate decisions is complicated by the fact that removing it compromises adhesion and Wnt signaling simultaneously, and sorting out which influence is critical is challenging. Valenta *et al.* [4], as reported in a recent issue of *Genes and Development*, describe a method to distinguish between βcat's roles in adhesion and Wnt signaling, which also provides insights into how the same transcriptional regulator regulates different suites of genes.

βcat binds TCF, the destruction complex, and E-cadherin using the same protein interaction surface — its central Armadillo (Arm) repeats (Figure 1A,B). However, βcat's amino and carboxyl termini are exposed for protein interactions [5]. At Wnt response elements, TCF binds DNA while βcat's amino- and carboxy-terminal regions recruit chromatin remodeling and basic transcription machinery (Figure 1C). The amino-terminal Arm repeats recruit both the Bcl9/Pygopus (Bcl9 = fly Legless) co-activator complex and Pontin 52, which links βcat to TATA-Box binding protein. Multiple proteins bind βcat's carboxy-terminal Arm repeats and unstructured region, including chromatin-modifying and remodeling factors like the histone acetyltransferases CBP/p300 and TRRAP-TIP60, as well as Brg1/Brahma and ISW1 [5]. βcat's carboxyl terminus also binds Parafibromin/Hyrax and MED12, which modulate transcriptional initiation and elongation by directly interacting with RNA polymerase II. Interestingly, fusing βcat's carboxyl terminus to TCF is sufficient to activate Wnt signaling [6]. βcat's carboxyl terminus also binds negative regulators of signaling like Chibby and ICAT. How the interplay of βcat's different interactors affects chromatin remodeling, transcription initiation and elongation is yet to be determined.

Previous work with fly βcat (Armadillo; Arm) revealed that one can generate mutations differentially affecting adhesion or signaling [7]. To explore which defects associated with loss of βcat are due to failure in Wnt signaling versus cell adhesion, Valenta *et al.* [4] designed mutations in fly and mammalian βcat specifically disrupting